peptidoglycan to gain access to the periplasm. Once there, the *Bdellovibrio* alters the shape of the prey cell, causing it to round up. Both of these processes clearly require hydrolytic enzymes and the importance of hydrolysis to the *Bdellovibrio* life cycle is underlined by the abundance of hydrolytic enzymes — a total of 293 are encoded, including 150 proteases and peptidases — and transporters — 244 open reading frames — in the genome.

Aside from being interesting as the 'world's smallest hunter', scientists are also interested in *Bdellovibrio* in the hope that this genus might be a source of novel enzyme-based antimicrobial compounds. The publication of the genome sequence could therefore potentially be the first step towards novel antibacterial strategies.

Sheilagh Clarkson

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VIROLOGY

HTLV-1 — showing some self-control

Sometimes, practising self-restraint can have its advantages. A recent study by Genoveffa Franchini, Christophe Nicot and colleagues provides an example of this, in which human T-cell lymphotrophic virus type 1 (HTLV-1) inhibits the expression of its own proteins — a strategy that could help it to avoid detection by the immune system.

p30^{II} — an HTLV-1 protein of previously unknown function — localizes to nuclei, and the authors therefore suspected that it might function in regulating viral gene expression. To investigate this, they co-expressed an HTLV-1 plasmid with a p30^{II} complementary DNA, and found that p30^{II} expression inhibited production of viral proteins in a dose-dependent manner.

The HTLV-1 Tax protein stimulates viral gene expression, so the authors investigated whether the effects of p30^{II} are mediated through inhibition of Tax-induced transcription. When p30^{II} was co-expressed with a Tax cDNA construct, there was no change in the transcription of Tax-dependent reporter genes. However, when Tax was expressed from a plasmid containing the full-length HTLV-1 sequence, p30^{II} inhibited transcription by Tax, indicating that p30^{II} might prevent the expression of Tax at the post-transcriptional level.

Using semiquantitative or real-time PCR on RNA from cells co-transfected with an HTLV-1 provirus and a p30^{II} construct, the authors found that p30^{II} expression had little effect on the levels of viral mRNAs in total cell extracts. However, when cytoplasmic RNA was analysed, there was a significant decrease in the level of the Tax/Rex mRNA, which contains sequences for both Tax and Rex — another positive regulator of viral gene expression. The Tax/Rex transcript, in contrast to other viral mRNAs, was detected in nuclear extracts, indicating that p30^{II} might bind specifically to this transcript and cause its retention in the nucleus.

The Tax/Rex and p21^{Rex} transcripts are produced by differential splicing of viral genomic RNA, and therefore contain different splice junctions. To confirm the specificity of the interaction between p30^{II} and Tax/Rex mRNA, Franchini, Nicot and colleagues made reporter constructs containing the splice junctions of either p21^{Rex} or Tax/Rex at their 3′ ends. Co-expressing p30^{II} inhibited expression from the construct containing the Tax/Rex splice junctions, but had no effect on the p21^{Rex} construct.

The authors then tested whether p30^{II} is able to move in and out of the nucleus. They transfected one set of cells with a plasmid expressing a red fluorescent protein (RFP) and another with a green-fluorescent-protein-tagged p30^{II} construct. When the two cell lines were fused, p30^{II} was unable to move into the nuclei of the RFP-expressing cells, unlike a control protein that shuttled between the nucleus and cytoplasm. This indicates how p30^{II} binding toTax/Rex mRNA might cause its nuclear retention, and therefore prevent its translation.

To confirm the inhibitory function of p30^{II} in a biologically relevant situation, the authors tested its ability to suppress the production of viral proteins in human T cells chronically infected with HTLV-1. Expression of p30^{II} from a recombinant lentivirus vector reduced the levels of viral proteins in supernatants from these cells.

By inhibiting the production of viral proteins, p30^{II} could enable HTLV-1 to avoid immune surveillance at key stages of infection — a strategy used by several other viruses. Whether this is the case, and if so, how the timing of p30^{II} activity is regulated will be important questions for future investigation.

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